A CASE REPORT ON AN ULCERATIVE PERIPHERAL GIANT CELL GRANULOMA

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ABSTRACT:

Peripheral Giant Cell Granuloma (PGCG) is a localized non-neoplastic lesion, characterized by reactive hyperplasia associated with plaque and calculus build up and any other local irritation and can also associated with oral trauma.

PGCG has a purplish to reddish appearance, presenting as a soft to firm exophytic growth with a pedicle or sessile base, and has a smooth surface which can be ulcerated. Histological analysis of PCGC demonstrated a polypoidal biopsy comprising squamous mucosa with ulceration. Within the stroma, there was an unencapsulated proliferation comprising numerous osteoclast-type giant cells embedded within a fibrous stroma with extravasation of red blood cells. This case report s on a PGCG diagnosed in the maxilla of an adult, female patient managed by surgical excision which had no recurrence.

Key Words: granuloma, irritation, ulcerative, excision.

INTRODUCTION:

Peripheral Giant Cell Granuloma (PGCG) was first described by Jaffe in 1953, and was originally called a reparative giant cell granuloma ^[1] is a localized non-neoplastic lesion. characterized bv reactive hyperplasia^[2]. It is commonly associated with poor oral hygiene associated with plaque and calculus build up and any other local irritation $\left[\frac{2-4}{4}\right]$ It is also associated with oral trauma from extractions, periodontal surgery and oral surgery $\left[\frac{2-4}{4}\right]$ In some case PGCG has been known to be associated with ill-fitting orthodontic appliances and defective restorations $\left[\frac{2-4}{2}\right]$.

PGCG lesions occur over a wide age range however some authors reported that they present mostly between the fourth and sixth decades of life ^[5,6] while other authors suggest that these lesions commonly occurs in males in the second decade and in females in the fifth decade ^[7,8]. PGCG lesions are found to occur twice as often in the mandible than in the maxilla^[8,9] and are thought to originate from periosteal or periodontal ligament cells ^[9]. PGCG lesions can be located in any region of the gingiva or alveolar mucosa^[2,10] in dentate or edentulous patients^[10-12]. Clinically, PGCG has a purplish to reddish appearance presenting as a soft to firm exophytic growth with a pedicle or sessile base, and has a smooth surface which can be ulcerated [6,13,14].

Differential diagnosis: It is imperative to recognize, diagnose, and treat any forms of oral pathology and PGCG must be differentiated from other similar lesions, such as pyogenic granuloma ^[2], traumatic fibroma, Browns tumour ^[1,2], peripheral

ossifying fibroma, and other similar lesions ^[2]. The treatment of a PCGC lesion consists of local surgical excision below the underlying bone and removal of any irritation agent in the region in order to minimize the risk of recurrence ^[8,14].

This case report describes the clinical , radiological and histopathological findings of a PGCG, confirmed by histopathological and radiological analysis, that was diagnosed in the maxilla of an adult, female patient. This case highlights the proper management of a PGCG lesion within a holistic patient oral care framework. The treatment of the lesion by surgical excision with curettage was a success, as the lesion showed no signs of recurrence after four years.

CASE DETAIL:

A healthy, 42 years old female patient presented with main complaint of pain associated with an ulcerative lesion on her palate. This lesion was located adjacent and anteriorly to the upper left third molar in the second molar position (Figure 1). She stated that the lesion had been progressively growing since she first noticed it as an irritation and assumed that it was a pimple on her palate, about 2 years ago. She tried to home therapy with rinsing with salt water which did not remove the growth instead it seemed to grow quite rapidly and become painful to the point of seeking care. The patient stated that she had extracted the upper left second molar about five vears ago without complications. The intraoral examination revealed a solitary nodular lesion

measuring approximately 20x15x8mm the palate anteriorly adjacent to the upper left third molar(28) in the missing second molar (27) position. The lesion was broad based , sessile and not pedunculated. On palpation, the lesion had a firm fibrous consistency and a smooth surface, except the top where it was ulcerated which could be attributed to occlusal trauma from the opposing retained roots of the 36 (<u>Figure 2</u>).



Figure 1. Solitary exophytic nodular lesion associated with the upper left third molar.



Figure 2. Ulceration on lesion associated with the upper left third molar.

The patient's panorex radiograph (Figure <u>3</u>) revealed that she had caries on the 14, 18 and 47, with peri-apical abscesses associated with the 14, 47 and retained roots of 36. The 25, 36 and 38 was retained roots, and the 21 had root canal treatment with a failed restoration. The

patient had generalised mild to moderate bone loss indicative of mild to moderate generalised periodontal disease. Failed restorations, plague and calculus was visibly present indicative of poor oral health status and poor oral hygiene (Figure 4). The upper left third molar (28) was found to be vital and had no lesion or periodontal involvement. The patient reported that as the lesion expanded she experienced pain whenever she bit or chewed hard and the lower retained roots pierced the lesion or when she ate something that caused irritation to the lesion. A full blood count was done and the results were found to be within normal limits.



Figure 3. Panorex radiograph reveals abscesses associated with carious teeth, retained roots and generalized bone loss.



Figure 4. Failed Restorations, inflamed marginal gingiva, plaque and calculus

indicating poor oral health status and oral hygiene

The patient's medical history revealed no systemic diseases, and the patient was not on any medication at the time. Extra oral examination revealed no significant findings.

Histo-pathological analysis

Histo-pathological analysis demonstrated a polypoidal biopsy comprising squamous mucosa with ulceration. Within the stroma, there was an unencapsulated proliferation comprising numerous osteoclast-type giant cells (Figure 5) embedded within a fibrous stroma with extravasation of red blood cells. The mitotic count was 3 per 10 at high power fields (Figure 6). There was haemosiderin, however there was no evidence of osteoid malignancy.The histo-pathological or features and the fact that there was no bone involvement, was indicative of a peripheral giant cell granuloma.

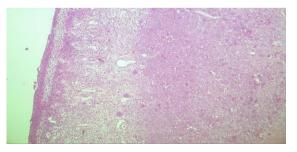


Figure 5. Nodular proliferation of cellular mesenchymal tissue with numerous multinucleated giant cells dispersed throughout at low magnification

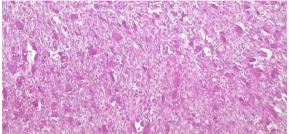


Figure 6. Higher magnification of PGCG showing unencapsulated proliferation comprising numerous osteoclast-type giant cells embedded within a fibrous stroma with extravasation of red blood cells.

Radiographic findings

Accurate classification of a PGCG depends on the radiological finding particularly if there is no bone involvement nor displacement of teeth. If the radiological findings indicate bone involvement, the differential diagnosis would include brown's tumour (hyperparathyroidism) and giant cell tumours of bone. In some superficial erosion of the instances interdental bone crest or of the alveolar bone margin in edentulous areas is noticeable.¹ No radiographic changes was observed except for the generalised bone loss indicative of generalised periodontitis which further asserted the diagnosis of PGCG (Figure 7).



Figure 7. Peri-apical radiograph revealed no pathological findings

Treatment

A comprehensive treatment plan was designed and explained thoroughly to the patient with the aim of initially improving the oral health condition and then excising the lesion thereafter constructing prosthesis to replace the missing teeth. This initially entailed oral hygiene instruction, antibiotic therapy to treat the oral abscess on the 47. The next appointment, a week later, oral hygiene instruction was given and root planning was done, and the peri -apically involved carious 14 and 47, retained roots of 25, 36 and 38 were all removed. The following week a scale and polish was done and the 21 was restored with the intention of placing a crown in future.

The lesion was monitored at each visit and at the next visit, a week later, the lesion was surgically excised down to the periosteum, which was curettaged, The excised tissue was placed in a container with formalin and sent for histopathological analysis.

The patient's first follow up visits was scheduled two week later which showed uneventful healing. There was closure of the excision site one month post operatively and two month later a crown on the 21, and a chrome denture to replace the missing teeth was constructed for the patient. Scale and polishing including oral hygiene instructions were performed at every recall visit. No signs of recurrence of

the lesion have been observed after two years post operatively and the patient was pleased with the results (Figure 8).



Figure 8. Clinical appearance after two years

DISCUSSION:

The origin of giant cells in PGCG has not been established though some authors concluded that the multinucleated cells in PGCG are of osteoclastic origin. derived from differentiated mononuclear cells^{[14},^{15]}. The mechanism that activates or recruits osteoclasts in PGCG has not yet been identified ^{13,14,15]}.

PGCG presents clinically as a firm, malleable, bright pedunculated or sessile nodule, generally less than 20 mm in diameter and be in the form of small papules to enlarged masses. the lesion is usually asymptomatic and its colour can be dark red to blue with or without an ulcerated surface [13,17,18] .This is similar to the lesion investigated which revealed a solitary nodular lesion measuring approximately 20x15x8mm. A PGCG is a reactive, non-neoplastic lesion caused by trauma or irritation. its presence is associated with poor oral hygiene, an

irritation or trauma^[7,13] or a combination of these as this case indicates.

The differential diagnosis

PGCG can be differentiated from lesions with very similar clinical and histological characteristics, by means of radiological evaluation^[17,18]. Central giant cell granulomas are more aggressive and located within the jaw itself while, PGCG and peripheral ossifying fibroma are lesions unique to the oral cavity, proliferative gingival lesions such as PG, gingival fibromatosis , peripheral ossifying fibroma, and are very similar clinically to PGCG, and may also be a response to chronic irritation characteristics but can present distinct infiltrative features and recurrence risk [11,1718] . PGCG is a soft tissue lesion and seldom affects the underlying bone, though the latter may suffer superficial erosion [11,17]

PGCG is treated with removal of source of irritation and surgical resection with curettage to ensure there is no recurrence $^{[14, 8, 25]}$. as was done for this case which resulted in no recurrence of the lesion and the last molar(38) was preserved Recurrence of PGCG is very seldom (5–11%) have been reported.^{4,19} occurrence of PGCG is very seldom in patients of 11–20 year old range from 6.5% to 12.7% $^{[3, 8]}$, while in children aged up to 10 years it is approximately 9% $^{[3, 8]}$.

The poor oral health status and poor oral hygiene of the patient affirm the belief that this lesion is commonly associated with poor oral hygiene associated with plaque

and calculus build up and any other local irritation^[2-4]. Some researchers have suggested that history of trauma might be related to the development of PGCG ^[11, 15] and in this case the trauma caused by the retained roots of the lower second molar (37) when biting or chewing, could be the traumatic factor that contributed to the development of the lesion,

PGCG can be diagnosed early based on clinical and radiological findings and can be followed through by proper conservative management which ensures minimized destruction for the adjacent teeth and tissues. A distinction between central and peripheral forms of giant cell granulomas^[15] will require radiological evaluation which is confirmed bv pathological analysis. Careful medical history followed by complete physical, immunological, and histopathological examination are critical procedures in the diagnosis process, aiming for a correct treatment plan and thereby reducing the possibility of recurrence and morbidity for patients.

CONCLUSION:

This case illustrates that the proper management of a PGCG lesion requires, proper diagnosis with the necessary radiological and histopathological investigations. Treatment should have a holistic approach aimed at removal of any cause of the lesion, surgical excision of the lesion with bone curettage, improvement of the oral health condition. This will ensure that the lesion does not recur as in this case, and hopefully improved oral health for the patient.

REFERENCES:

- Jaffe H. L (1953). Giant-cell reparative granuloma, traumatic bone cyst, and fibrous (fibro-osseous) dysplasia of the jawbones. Oral Surgery, Oral Medicine, Oral Pathology, vol. 6, no. 1, pp. 159–175,. View at Publisher · View at Google Scholar · View at Scopus
- Lester S. R, Cordell K. G, Rosebush M. S, Palaiologou A. A, Maney P (2014). Peripheral giant cell granulomas: a series of 279 cases. Oral Surgery, Oral Medicine, Oral Pathology and Oral Radiology, vol. 118, no. 4, pp. 475– 482,. View at Publisher · View at Google Scholar · View at Scopus
- Shadman N, Ebrahimi S. F, Jafari S, Eslami M (2009). Peripheral giant cell granuloma: a review of 123 cases. *Dental Research Journal*, vol. 6, no. 1, pp. 47–50, View at Google Scholar
- Mighell A. J, Robinson P. A, Hume W. J (1995). Peripheral giant cell granuloma: a clinical study of 77 cases from 62 patients, and literature review, Oral Diseases, vol. 1, no. 1, pp. 12–19, View at Google Scholar · View at Scopus
- 5. Motamedi M. H. K, Eshghyar N., Jafari S. M. et al., (2007). Peripheral and central giant cell granulomas of the Demographic Study, Oral jaws: A Surgery, Oral Medicine. Oral Pathology, Oral Radiology and Endodontology, vol. 103, no. 6, pp. e39-e43, View at Publisher · View at Google Scholar · View at Scopus
- Zarei M. R, Chamani G, Amanpoor S (2007). Reactive hyperplasia of the oral cavity in Kerman province, Iran: a

review of 172 cases, British Journal of Oral and Maxillofacial Surgery, vol. 45, 288-292, 4, pp. View no. at Publisher · View Google at Scholar · View at Scopus

- 7. Soames J.V, Southam J.C (2000). Oral pathology; 4th ed. Oxford. [Google Scholar]
- 8. Katsikeris N, Kakarantza-Angelopoulou E, Angelopoulos A.P (1988). Peripheral giant cell granuloma: clinicopathologic study of 224 new cases and review of cases. Int J Oral 956 reported Surg. ;17:94-Maxillofac 99. [PubMed] [Google Scholar]
- 9. Chaparro Avendaño A. V, Berini Aytés L, Escoda C. G (2005). Peripheral giant cell granuloma. A report of five cases and review of the literature, Medicina Oral, Patologia Oral y Cirugia Bucal, vol. 10, no. 1, pp. 48-57, View at Google Scholar · View at Scopus
- 10. Nekouei A, Eshghi A, Jafarnejadi P, Enshaei Z (2016). A Review and Report of Peripheral Giant Cell Granuloma in a 4-Year-Old Child, Case Reports in Dentistry, vol., Article ID 7536304, 4 pages,

2016. https://doi.org/10.1155/2016/7 536304

- 11. Flaitz C.M (2000). Peripheral giant cell granuloma: a potentially aggressive lesion in children. Pediatr Dent. ;22:232-233. [PubMed] [Google Scholar]
- 12. Kfir Y, Buchner A, Hansen L (1980). Reactive lesions of the gingiva: a clinicopathological study of 741 cases. J Periodontol.;51:655-661. [PubMed] [Google Scholar]
- 13. Bodner L, Peist M, Gatot A, Fliss D. M (1997). Growth potential of peripheral giant cell granuloma. Oral Surg Oral Med Oral Pathol Oral Radiol Endod.;83:548-

551. [PubMed] [Google Scholar]

- 14. Brown A. L, de Moraes P. C, Sperandio M, Soares A. B, Araújo V. C, Passador-Santos F (2015). "Peripheral giant cell granuloma associated with a dental implant: a case report and review of literature, Case the Reports in Dentistry, vol. 2015, Article ID 697673, 6 pages, View at Publisher View at Google Scholar · View at Scopus
- 15. Abu Gharbyah A. Z , Assaf M (2014). Management of a peripheral giant cell granuloma in the esthetic area of upper jaw: а case report, International Journal of Surgery Case Reports, vol. 5, no. 11, pp. 779-782, View at Publisher · View at Google Scholar · View at Scopus
- 16. Sahingur S. E, Cohen R. E, and Aguirre A (2004). Esthetic management of peripheral giant cell granuloma, Journal of Periodontology, vol. 75, no. 3, pp. 487-492, View at Publisher · View Google at Scholar ·View at Scopus
- 17. Pandolfi P.J, Felefli S, Flaitz C.M, Jhonson J.V (1999). An aggressive peripheral giant cell granuloma in a child. J Clin Pediatr Dent. ;23:353-355. [PubMed] [Google Scholar]
- 18. daSilva F. C, Piazzetta C. M, Torres-Pereira C. C, Schussel J. L, Amenábar J. M (2016). Gingival proliferative lesions in children and adolescents in Brazil: a 15-year-period crosssectional study, Journal of Indian Society of Periodontology, vol. 20, no. 1, pp. 63-66,. View at Publisher · View at Google Scholar